sures rely upon basal body temperature charts, cervical mucus examination and previous menstrual history as guides. In some cases, hormone assays like rapid estradiol or luteinizing hormone have been of great use; even when all of these measures are used accurately, however, predicting the time of ovulation can be a significant clinical problem. Because the viable lifespan of a human ovum is 16 to 18 hours and preceding fertilization the sperm must undergo a four- to six-hour transformation termed capacitation, precise timing becomes critical.

This problem has been substantially eliminated in the past two years through in vitro fertilization programs and application of real-time ultrasound as a means of timing laparoscopic recovery of ova. Using a full-bladder technique, ovaries and their developing follicles can be readily defined in transverse and longitudinal planes. Follicular growth proceeds at a rate of 1 to 3 mm a day, with a rapid growth spurt possible some 24 to 36 hours before ovulation. Follicles are considered to be functionally mature when their maximal diameter is 18 mm or more. Follicles of this size will respond to ovulation doses of human chorionic gonadotropin (5,000 to 10,000 IU) within 12 to 36 hours after intramuscular injection.

Ultrasound used in this manner does not replace more conventional measures of impending ovulation, but it does hold the promise of better understanding of a given patient's ovarian response to natural or drug-induced signals. Completed studies suggest that its use increases pregnancy rates while shortening course of treatment.

PAUL S. WEATHERSBEE, PhD

REFERENCES

DeCherney AH, Romero R, Polan ML: Ultrasound in reproductive endocrinology. Fertil Steril 1982 Mar; 37:323-333

Kerin JF, Edmonds DK, Warnes GM, et al: Morphological and functional relations of graafian follicle growth to ovulation in women using ultrasonic, laparoscopic and biochemical measurements. Br J Obstet Gynaecol 1981 Feb; 88:81-90

Orsini LF, Rizzo N, Calderoni P, et al: Ultrasound monitoring of ovarian follicular development: A comparison of real-time and static scanning techniques. JCU 1983 May; 11:207-213

Changing Views on the Management of Vaginitis

THERE HAVE BEEN a number of significant advances in the treatment of infectious vaginitis in the past five years, not only through the introduction of new drugs but also by using existing drugs for new indications. In view of the fact that the three major types of vaginitis are all to some extent sexually transmitted, it is likely that the number of women seen with complaints referable to vaginitis will continue to increase.

The most common cause of infectious vaginitis is *Gardnerella vaginalis* vaginitis, previously called *Haemophilis vaginalis* vaginitis or nonspecific vaginitis. The infection is characterized by a profuse, homogeneous, adherent gray discharge that is associated with a "fishy" odor and a minimum of irritative symptoms. Although the laboratory diagnosis has relied on recognition of "clue cells" in the saline wet mount, another helpful diagnostic maneuver is the "odor" or "amine" test in which the odor of the discharge worsens upon adding 10% potassium hydroxide solution to the residue on the posterior blade of the speculum. Treatment has traditionally consisted of topically applied sulfa creams or oral tetracycline, but it is now generally agreed that these measures are relatively ineffective and that the agent of choice is metronidazole, 500 mg given twice a day for seven days to both the patient and her partner. In patients in whom metronidazole is contraindicated (such as pregnant women), cephalexin or ampicillin, 500 mg given four times a day for seven days, are secondary drugs of choice. A contrary opinion was recently given by Robbie and Sweet when they reported their reservations regarding the extensive and repetitive use of metronidazole because of its possible mutagenicity and carcinogenicity when more innocuous methods are available. They suggest that local preparations or ampicillin be used as the treatment of choice and that metronidazole be reserved for initial treatment failures.

Women with monilial vaginitis usually have vaginal and vulvar itching and burning and a nonodorous white vaginal discharge. Diagnosis is made on the basis of identification of pseudohyphae on a 10% KOH slide or with a positive yeast culture. Nystatin, which had previously been the drug of choice, has now been replaced by the more effective synthetic imidazole fungicides, clotrimazole and miconazole nitrate, which are available as creams, vaginal tablets and suppositories. Although standard therapy consists of a daily application for seven days, a recent study has shown equivalent results with twice-a-day therapy for three days, thereby giving patients a choice of regimens.

Trichomonas vaginalis vaginitis presents as vaginal itching and burning in conjunction with a foul-smelling watery discharge. Diagnosis depends on the identification of motile trichomonads on saline wet prep or a positive Diamonds culture. The mainstay of treatment continues to be metronidazole, though recent studies have shown that a 2-gram single dose is as effective as the more standard seven-day course. Because of improved compliance and minimization of the duration of side effects, most authors consider the single-dose regimen, given to both patient and partner, to be the treatment of choice for vaginal trichomonas.

MICHAEL S. POLICAR, MD

REFERENCES

Centers for Disease Control: Sexually transmitted diseases treatment guidelines 1982. MMWR 1982 Aug 20; 31(suppl):33S-60S

Fleury FJ: Adult vaginitis. Clin Obstet Gynecol 1981 Jun; 24:407-438

Gardner HL: Haemophilus vaginalis vaginitis after twenty-five years. Am J Obstet Gynecol 1980 Jun 1; 137:385-391

Lebherz TB, Ford LC, Kleinkopf V: A comparison of a three-day and seven-day clotrimazole regimen for vulvovaginal candidiasis. Clin Ther 1981; 3(5):344-348

Robbie MO, Sweet RL: Metronidazole use in obstetrics and gynecology: A review. Am J Obstet Gynecol 1983 Apr 1; 145:865-881

Glucocorticoids and Lecithin-Sphingomyelin Ratio

THE ANTENATAL USE of glucocorticoids to prevent respiratory distress syndrome in premature infants has accelerated over the past several years. The rationale for their use is based on several studies done of humans and animals that have shown the following: (1) Exogenously administered steroids accelerate the normal pattern of lung development, including the accumula-

tion of lamellar bodies in type II cells and the production of alveolar surfactant. In humans, glucocorticoids decrease the incidence of respiratory distress syndrome and increase survival rate for premature infants. (2) Most likely increasing concentrations of endogenous corticosteroids in an undisturbed or a stressed fetus influence lung development in the same manner as exogenously administered steroids. (3) When exogenously administered glucocorticoids are used in dosages that do not elevate fetal cortisol concentrations beyond that seen with physiologic stress responses, they present little risk to an infant. (4) Morphologic, functional and biochemical variables of lung development are more closely correlated to cortisol concentrations than is fetal age.

Although the lecithin-sphingomyelin ratio in amniotic fluid has proved to be a reliable index of fetal lung maturity, the amniotic fluid "lung profile" can more accurately predict fetal lung maturation in humans. Several studies have shown an increase in lecithin-sphingomyelin ratio after exogenous glucocorticoid administration, whereas others failed to do so, albeit noting a significant reduction in the incidence of respiratory distress syndrome and increased fetal survival. The latter findings suggested that either adequate amounts of surfactant should be present in alveoli before their appearance in amniotic fluid or that qualitative alterations in surfactant composition not reflected by the lecithin-sphingomyelin ratio have occurred. Recent findings in alloxan-treated animals show that exogenously administered glucocorticoids increase phosphatidylglycerol synthesis and reverse the functional delay in fetal lung maturity, with no significant change in surfactant content or the lecithin-sphingomyelin ratio. The above findings attest to the added advantage of using an amniotic fluid "lung profile" specifically in those clinical situations—such as a case of diabetes mellitus with no vascular involvement and Rh immunization—wherein delayed lung maturity is expected.

Studies of surfactant metabolism show that maximal beneficial effects of glucocorticoids are obtained after 36 hours of administration. Also, for continued benefit, the same dose should be repeated seven to ten days later. Of great interest are the recent observations that fetal responses to glucocorticoid administration are influenced by sex and ethnic origin.

I recommend that the use of glucocorticoids for fetal lung maturity be limited to women in premature labor whose amniotic fluid analysis is not available, or is in the immature range, and whose labor can be delayed for at least 36 hours; where immediate delivery is indicated (that as, in cases of severe hypertension or abruptio placenta), glucocorticoid administration is contraindicated. More studies are needed to define optimal mode of drug delivery, dosage, short-term maternal risks and the long-term effects on neonatal central nervous system and immune systems.

BAHIJ NUWAYHID, MD

REFERENCES

King RJ: Pulmonary surfactant. J Appl Physiol 1982 Jul; 53:1-8

Teramo K, Hallman M, Raivio KO: Maternal glucocorticoid in unplanned premature labor—Controlled study on the effects of betamethasone phosphate on the phospholipids of the gastric aspirate and on the adrenal cortical function of the newborn infant. Pediatr Res 1980 Apr; 14:326-329

Tsai MY, Josephson MW, Donhowe J: Delayed pulmonary phosphatidylglycerol synthesis and reversal by prenatal dexamethasone in fetal rats of streptozotocin diabetic mothers. Exp Lung Res 1983 May; 4:315-323

Immunologic Thrombocytopenia in Pregnancy: Predicting Fetal-Neonatal Risks

IMMUNOLOGIC THROMBOCYTOPENIC PURPURA is a relatively uncommon disorder whose occurrence in the general population includes a significant number of women of childbearing age. As a result, obstetricians can be faced with the problem of managing such a patient during a pregnancy.

Earlier reports had suggested an increase in maternal mortality to rates as high as 14%. Similarly, perinatal death rates of 15% to 25% had also been reported, related at least in part to thrombocytopenia in neonates. This background information, as well as the absence of any more sensitive laboratory evaluation than maternal platelet count, probably contributed to the recommendation of a "prophylactic" cesarean section when the maternal platelet count was less than 100,000 per μl. More recent studies, however, that examined the immunologic mechanisms of this disorder and their application in pregnant patients, have resulted in a more selective use of cesarean section in such patients.

Immunologic thrombocytopenic purpura is caused by antiplatelet antibody of the subclass 3 of immunoglobulin G. As well, there appear to be placental receptors for the Fc portion of the IgG molecule that can account for active transport of antibody to the fetal circulation. The level of antiplatelet antibody can be measured in maternal serum with respect to both free circulating antibody and bound, or platelet-associated, antibody. In addition, the latter techniques can still be used whether a patient is in remission or has active disease, and whether or not she is receiving steroids.

In 1982 Cines and co-workers examined levels of free and associated antiplatelet antibody in 23 pregnant women. While the maternal levels of platelet-associated antibody did not correlate with thrombocytopenia in their neonates, the levels of circulating antibody were elevated in 11 women, 9 of whom had thrombocytopenic neonates (defined as having a platelet count of less than 150,000 per μl). Further, the mothers who had the highest levels of circulating antibody had the more severely affected neonates (a platelet count of less than 30,000 per μl).

In 1983 Scott and associates similarly examined circulating antiplatelet antibody levels. While they found thrombocytopenic neonates in mothers with elevated antibody levels, they also noted affected neonates in mothers whose levels were normal. They therefore also used fetal scalp blood platelet counts in 25 patients and correctly identified the five thrombocytopenic in-